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Re: El-Garem et al.: Seminal *Helicobacter pylori* Treatment Improves Sperm Motility in Infertile Asthenozoospermic Men (Urology 2014;84:1347-1350).

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On *Helicobacter pylori* treatment: involvement in male infertility

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We have read the paper by El-Garem et al reporting the effect of *Helicobacter pylori* (*H. pylori*) treatment on sperm motility in infertile asthenozoospermic men.¹ Evaluating seminal antibodies (IgA) to *H. pylori* by enzyme-linked immunosorbent assay on a total of 223 men with progressive and nonprogressive sperm motility, authors found elevated levels of seminal *H. pylori* IgA antibody in 22 (9.8%) subjects. These cases were treated with anti-*H. pylori* triple therapy based on omeprazole, tinidazole and clarithromycin. After treatment, a significant decrease of antibody levels was observed in association with a significant increase in sperm motility and normalization of morphology. Considering a potential antigenic mimicry mechanism, inducing an autoimmune cross-reaction between antibody directed to bacterial antigens and β -tubulin protein of human spermatozoa, authors concluded that *H. pylori* treatment improved sperm motility suggesting the research of seminal antibodies to *H. pylori* in asthenozoospermic men.¹

H. pylori is a Gram-negative bacterium, its niche is the stomach where can cause gastritis and peptic disease. Although since two decades extra-gastric manifestations have been attributed to this bacterium but only for a small part of these a role has been proved.²

Although the authors correctly reported the lack of association between serum and seminal IgA, a clear clinical message should be highlighted. In this study the presence of *H pylori* has not been shown because the assessment of antibodies to the bacterium in semen is not an appropriate test. In fact, according to International guidelines the non-invasive diagnosis of *H pylori* infection should be based on either ¹³C-Urea breath test (sensitivity 88-95% and specificity 95-100%) or monoclonal stool antigen test (sensitivity 94% and specificity 92%), called direct tests; as last resort, only validated IgG serology should be used (indirect test).³ Indeed, the presence of antibodies to *H pylori* could represent just a marker of previous exposure without necessarily indicating a current infection. In the context of interventional studies the use of a direct diagnostic test both prior to and after treatment is mandatory.

Since the message highlighted in this paper¹ could carry relevant implications in clinical practice, we suggest that future studies on this issue should use appropriate and validated methods for *H pylori* diagnosis in order to avoid unnecessary antibiotic treatments in potential uninfected individuals.

References

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